> they reported finding a significant interaction between social class and paternal smoking effects, and earlier studies had reported interactions between effects associated with maternal and paternal smoking status. Rubin and coworkers failed to control for a number of other potential confounders as well.[10]

A study which reports the following is also cited by OSHA:

> We report <u>negative results</u> on induction of chromosomal damage in 2 separate groups of intensive involuntary exposure to tobacco smoke, non-smoking restaurant personnel and newborn children of smoking mothers. (Ex. 8-287) [emphasis added]

Finally, the other study cited by OSHA is a paper on the possible effects of active smoking by the mother during pregnancy. (Ex. 8-299) The paper contains no data on pregnant mothers who are exposed to ETS in the workplace during pregnancy.

OSHA fails to discuss a number of other studies which have reported no statistically significant associations between maternal exposure to ETS during pregnancy and the delivery of low birthweight infants.[13-19] In fact, one study considered 57 different risk factors for low birthweight infants and reported that paternal smoking (as an index of exposure to ETS) had no statistically significant effect on infant birth-weight.[13]

OSHA does not provide a detailed discussion of the clinical significance, if any, of the reported decrements in infant birthweight allegedly associated with ETS exposure of the mother during pregnancy.

OSHA'S CLAIM THAT PRENATAL EXPOSURE TO ETS AND EXPOSURE TO ETS AS A CHILD MAY INCREASE AN INDIVIDUAL'S CANCER RISK BY A FACTOR OF TWO IS UNSUPPORTED BY A THOROUGH REVIEW OF THE AVAILABLE LITERATURE

In an attempt to substantiate its claim regarding the possible risk of cancer in nonsmokers exposed either prenatally or as a child to ETS, OSHA cites three papers. (Exs. 8-65, 8-164, 8-252) The first study reported an association between exposure to maternal smoking during childhood and an increase in lung cancer risk in nonsmokers in only one subgroup of subjects. Janerich et al., 1990, reported only one statistically significant risk estimate out of 13 exposure categories. The third reference is a reply to a Letter to the Editor regarding the Pershagen 1987 study (which itself is not referenced by OSHA). (Ex. 8-252) The Pershagen study reported "no consistent evidence of an effect," and the 95% confidence interval encompassed 1.0 for both histologic groups. Therefore, the studies' risk estimates were not statistically significant.

The two studies and the Letter to the Editor cited by OSHA examine the potential effect on an individual's cancer risk from exposure to parental smoking during childhood. These studies are therefore irrelevant to a discussion of potential health effects on the fetus from exposure of the mother to ETS in the workplace during her pregnancy. Nonetheless, a reviewer of this literature concluded that "the data do not indicate any association at all between risk of lung cancer in never smokers and exposure to ETS at work, or in childhood."[20]

OSHA PROVIDES A REFERENCE TO ONE EXPERIMENTAL ANIMAL STUDY WHICH IT CLAIMS IS SUPPORTIVE OF THE CONTENTION THAT EXPOSURE OF PREGNANT MOTHERS TO ETS IN THE WORKPLACE PRESENTS A SIGNIFICANT RISK OF MATERIAL HEALTH IMPAIRMENT TO THE FETUS

Although OSHA acknowledges that "experimental research on the adverse reproductive effects associated with ETS exposure in animals is limited," they do report on one study. (Ex. 8-6) This study reported that sciatic nerve tissue taken from the offspring of fresh sidestream smoke-exposed female mice revealed "definite toxic effects" on the neonatal tissue. There are several limitations of this study. Probably the most important is the small sample size. The authors reported data on only six exposed offspring and six controls. In addition, COHb levels of the exposed pregnant mice were 9%, which the authors report is

"equivalent to that found in humans who actively smoke 10-20 cigarettes per day." As one author suggested, studies which simulate mainstream smoke and employ levels equal to or greater than those expected from active smoking generate data which "are not appropriate for assessment of the likelihood or nature of ETS effects." The author also suggested that the authors of such studies should realize that "careful attention must be given to ensure that observed effects are not simply the result of maternal toxicity and systemic stress resulting from excessive doses."[10]

The authors of this study themselves also noted that these reported changes have been observed in other studies, including studies of the diabetic strain of the C57BL/KsJ mouse and in studies of decreased maternal food intake. They concede that "the irregularities noted in our investigation could be attributed to causes other than cigarette smoke inhalation as previous studies demonstrate."

REFERENCES

1. Windham, G., et al., "Parental Cigarette Smoking and the Risk of Spontaneous Abortion," American Journal of Epidemiology 135(12): 1394-1403, 1992.

2. Pan, M., et al., "The Influence of Passive Smoking on the Fetus During Pregnancy," China Obstetrics and Gynecology Magazine 6(27): 1-9, 1992.

3. Savitz, D., et al., "Influence of Paternal Age, Smoking, and Alcohol Consumption on Congenital Anomalies," Teratology 44: 429-440, 1991.

4. Tikannen, J. and Heinonen, O., "Risk Factors for Atrial Septal Defect," Eur J Epidemiology 8(4): 509-515, 1992.

5. Tikannen, J. and Heinonen, O., "Occupational Risk Factors for Congenital Heart Disease," International Archives of Occupational and Environmental Health 64: 59-64, 1992.

6. Ekwo, E., et al., "Risks for Premature Rupture of Amniotic Membranes," International Journal of Epidemiology 22(3): 495-503, 1993.

7. Hakim, R. and Tielsch, J., "Maternal Cigarette Smoking During Pregnancy: A Risk Factor for Childhood Strabismus," Arch Ophthalmol 110: 1459-1462, 1992.

8. Erickson, J., "Risk Factors for Birth Defects: Data from the Atlanta Birth Defects Case-Control Study," Teratology 43(1): 41-51, 1991.

9. Cnattingius, S., et al., "Who Continues to Smoke While Pregnant?," British Medical Journal 297: 258-261, 1992.

10. Hood, R.D., "As Assessment of Potential Effects of Environmental Tobacco Smoke on Prenatal Development and Reproductive Capacity," Environmental Tobacco Smoke Part VI: Proceedings Int Symp Environmental Tobacco Smoke, 1990.

11. Olsen, J., and Frische, G., "Social Differences in Reproductive Health," Scand J Soc Med 21(2): 90-97, 1993.

12. Trichopoulos, D., "Passive Smoking, Birthweight, and Oestrogens," Lancet II: 743, 1986.

13. Rantakallio, P., et al., "The Effect of Parental Smoking and Industrial Pollution on Birth Weight," Indoor Air Quality,

ed., H. Kasuga, Berlin, Heidelberg, Springer-Verlag: 219-225, 1990.

14. Little, R. and Sing, C., "Father's Drinking and Infant Birth Weight: Report of An Association," Teratology 36: 59-65, 1987.

15. Chen, Y., et al., "Passive Smoking and Low Birthweight," Lancet II: 54-55, 1989.

16. Kikuchi, S. and Takahashi, T., "Effect of Paternal Smoking on Fetuses," Indoor Air Quality, ed., H. Kasuga, Berlin, Heidelberg, Springer-Verlag: 272-277, 1990.

17. Ogawa, H., et al., "Passive Smoking by Pregnant Women and Fetal Growth," Journal of Epidemiology and Community Health 45: 164-168, 1991.

18. Fortier, I., et al., "Passive Smoking During Pregnancy and the Risk of Delivering a Small-for-Gestational-Age Infant," American Journal of Epidemiology 139(3): 294-301, 1994.

19. MacArthur, C. and Knox, E., "Passive Smoking and Birthweight," Lancet I(8523): 37-38, 1987.

20. Lee, P.N., Environmental Tobacco Smoke and Mortality, Karger Publishing, New York, New York, 1992.

SECTION X

MATERIAL IMPAIRMENT: GENOTOXICITY

GENOTOXICITY

OSHA FAILS TO JUSTIFY ITS INCLUSION OF GENOTOXICITY AS A SEPARATE CATEGORY UNDER THE MAJOR HEADING OF "HEALTH EFFECTS"; OSHA FAILS TO SHOW THAT GENOTOXICITY IS A MATERIAL IMPAIRMENT TO HEALTH

Genotoxicity refers to damage to DNA (the hereditary material). Permanent, heritable changes in the DNA are called mutations, and may be examined in tests for mutagenicity, such as the Ames test, in which mutation rates are measured in a particular strain of bacteria. Some correlations have been made between mutagenicity and carcinogenicity (the ability to induce cancer).

Other tests used to assess genotoxicity include the sister chromatid exchange (SCE) assay, which measures the frequency of exchange of portions of genetic material between two identical strands of DNA, and the chromosome aberration (CA) assay, which measures structural irregularities in strands of DNA. DNA adducts, abnormal configurations or clumping of the genetic material, may also be examined.

All forms of life are constantly exposed to physical and chemical agents in the environment (e.g., radiation) and to endogenous (internal) agents with the ability to cause changes in DNA. DNA has been called an "unstable" molecule, and it has been

noted that endogenous DNA damage may occur at the rate of 100,000 base pairs per cell, per day.[1,2]

According to Bruce Ames, developer of the Ames assay for mutagenicity, human exposure to potentially mutagenic or carcinogenic substances is much greater than generally appreciated, e.g., the environment can be thought of as "filled with potential carcinogens."[2]

Moreover, conclusions about genotoxicity obtained from in vitro systems, while certainly providing some information about the substance being tested, must nevertheless be put into the proper biological context. The magnitude of a genotoxic response in the whole organism may be substantially different than that observed in a bioassay. As Ames and Gold noted[3]:

> [H]umans have numerous inducible defense systems against mutagenic carcinogens, such as DNA repair, antioxidant defenses, glutathione transferases, and so forth . . . [L]ow doses of carcinogens appear to be both much more common and less hazardous than is generally thought.

Given the ubiquity of mutagens in the environment and the existence of numerous "defense systems" against mutagens, OSHA has not provided sufficient information in the Proposed Rule to support

its treatment of genotoxicity as a "health effect" or to establish genotoxicity as a material impairment to health.

OSHA'S REVIEW OF THE LITERATURE ON THE CLAIMED GENOTOXICITY OF ETS IS INCOMPLETE; A REVIEW OF ADDITIONAL RELEVANT STUDIES PROVIDES NO SUPPORT FOR OSHA'S CLAIM

OSHA's discussion of genotoxicity begins with a series of statements about the claimed correlations between genotoxicity and carcinogenicity. (Section II.C.7., 59 FR 15981) OSHA fails to provide references in support of these statements, although they appear to be OSHA's justification for discussing genotoxicity.

The Proposed Rule then contains a discussion of a number of studies in which cigarette smoke or cigarette smoke condensate was tested in the Ames Salmonella typhimurium assay, and an increased mutation rate was reported. OSHA's inclusion of studies dealing with mainstream and sidestream smoke again reveals the misconception pervading the Proposed Rule that ETS, mainstream, and sidestream smoke are equivalent.

OSHA omitted at least one relevant study from this discussion in the Proposed Rule. In 1991, Bombick, et al., reported on a cellular smoke exposure techniques using rat liver cells and the Ames Salmonella assay. After a three-hour exposure

using ETS at a concentration of 1.5 mg total particulate matter/m^3, the authors report[4]:

> Using the neutral red cytotoxicity and Ames mutagenesis assays there were no differences observed in the ETS-exposed cells and their respective room air controls, indicating that ETS was biologically inactive as tested. [emphasis added]

The Proposed Rule also discusses studies reporting that various constituents and extracts of ETS collected from indoor air are capable of inducing mutations in the Ames assay are also discussed. (Exs. 4-202, 4-5, 4-198, 4-201, 4-203) However, the significance of such reported findings has not been established. Virtually all air samples, whether in the presence or absence of smoking, can be shown to be mutagenic in various bioassays. Indeed, no substance, including foods and natural materials, has been unequivocally shown to be free of mutagenic and/or carcinogenic properties.

Of relevance, a study not cited by OSHA has reported that sidestream smoke exhibits reduced activity as it ages and becomes diluted, that is, as it becomes ETS.[5] Sonnenfeld and Wilson report on an experiment in which cultured mouse fibroblast-like cells were exposed to mainstream or sidestream smoke of various ages. In

this report, cytotoxicity (cell mortality) is used as a measurement of DNA damage sufficient to cause cell death. The authors write:

> Aging of SS smoke resulted in a rapid decline in the mortality generated by the smoke. As calculated from the linear regression curve, an increase in age of SS smoke of 30 [seconds] after generation would have resulted in a total loss of cytotoxic effects. [emphasis added]

The Proposed Rule almost completely overlooks genotoxicity studies using bodily fluids of persons exposed to ETS; these data do not allow a conclusion about genotoxicity to be reached

One area of research essentially overlooked in OSHA's Proposed Rule comprises those studies that have compared the mutagenicity of body fluids of nonsmokers exposed to ETS and nonsmokers not exposed to ETS. Several of these studies report no significant difference in mutagenic activity.[6-10] (Ex. 8-152)

For instance, in research by a team of German researchers, ten nonsmokers were exposed to ETS, generated by human smokers, for eight hours under two exposure conditions.[9] The two experiments were characterized by CO levels of 10 ppm and 25 ppm, respectively; according to the authors, both exposure regimes represent higher exposures than "real-life" situations. Elsewhere,

they described Experiment 2 as "far from being realistic,"[6] and bearing "no relation to a real-life situation."[8] In addition, the authors controlled for the effect of mutagens from the diet by keeping their subjects on a diet low in polycyclic aromatic hydrocarbons. Urine samples from both smokers and nonsmokers were tested in the Ames Salmonella assay. The authors report:[9]

> All urine extracts of ETS exposed non-smokers were found to be negative in the mutagenicity test when applying the [criterion] of Ames (doubling of spontaneous mutation rate).

Thus, even at exposure levels higher than would be expected on average, no increase in mutagenicity could be measured. These data do not support claims that ETS exposure is associated with an increase in mutagenic activity.

Citing the high variability of measures of urinary mutagenicity and questions about the relevance of increased urinary mutagenicity to cancer risk, the authors write:[8]

> These considerations lead to the conclusion that measuring the urinary mutagenic activity, at least in passive smokers, is not an appropriate method of predicting an increased risk to human health.

The authors also say:[8]

> The data suggest that nonsmokers in real-life situations take up very low doses of ETS constituents, and detoxification of the genotoxic substances inhaled is effective.

And:[10]

> Whether ETS exposure can lead to an elevated urinary mutagenicity is a matter of controversy. In most investigations no significant increase has been observed. . . .
>
> In our experience, the Ames test for detection of urinary mutagenicity is far too insensitive to assess such a low exposure. The results of our investigations, as well as those of other authors, suggest that urinary mutagenicity, which would be a potential marker for ETS particle exposure, remains unchanged after ETS exposure.

The few studies reporting statistically significant increases in urinary mutagenicity among individuals exposed to ETS did not employ realistic levels of exposure to ETS, and they did not control adequately for the presence of mutagens in the diet of the study subjects.[11-13] For instance, in the Bos, et al., study, the exposure condition consisted of the smoking of 157 cigarettes over six hours in a room with "poor ventilation."[11] The relevance of such an exposure to "real-life" conditions is certainly questionable. With respect to diet, Bartsch, et al., acknowledge, concerning their study, that[13]

Urinary mutagenicity is influenced also by dietary habits; although we collected information on diet, the dimension of the study (particularly as far as passive smokers are concerned) does not allow adequate statistical treatment of this potential confounding factor.

Other related studies have examined levels of various DNA changes in nonsmokers exposed to ETS.[1,14-17] (Exs. 8-152, 8-287) Based on the data presented in these studies, nonsmokers exposed to ETS do not appear to exhibit increased DNA adduct formation, nor do studies report increased levels of chromosomal changes in cells of nonsmokers exposed to ETS. Discussion of these studies follows.

Collman, et al., collected data from 16 nonsmokers, 15 "passive smokers" (currently living with one or more smokers), and 13 current smokers, all women.[14] Sister-chromatid exchange (SCE) frequencies in lymphocytes (a type of white blood cell) were compared with and without coincubation with a chemical that enhanced the frequency of SCEs. Based on both assays, the authors report that "the frequency of SCEs in persons passively exposed to smoke was not higher than in nonsmokers."

In a report by Husgafvel-Pursiainen, peripheral blood lymphocytes were examined for SCE frequency.[15] Study groups consisted of 12 smoking waiters and waitresses, 20 nonsmoking waiters and waitresses who were occupationally exposed to ETS, and

14 nonexposed office workers. The author reports that "[t]he mean SCE level in exposed non-smokers did not differ from that observed in the non-exposed group." Although no ETS measurements from the restaurants were reported, the author characterizes them as "heavily polluted," and the exposure as "long-term." This study, which reports data from persons exposed in a "real-life" situation, does not support claims of the genotoxicity of ETS.

Chromosomal aberrations (CAs) and SCEs were examined in peripheral blood lymphocytes from nine smoking waiters, 16 nonsmoking waiters exposed to ETS at work, and 7 reportedly nonexposed nonsmokers by Sorsa, et al. (Ex. 8-287) The authors report that "[n]o significant differences were seen between the groups or subgroups in the 2 parameters." Thus, no "genotoxic" effects could be detected in persons exposed to ETS at "real-world" levels.

Holz, et al., report that DNA adduct levels were compared in monocytes (a type of white blood cell) of smokers and "heavily exposed passive smokers," who had been exposed in a chamber.[16] DNA adducts above background were reported in smokers; they disappeared in less than 40 hours. The authors report no above-background adduct levels in study subjects exposed to ETS.

In a study by Gorgels, et al., 50 self-reported ETS-exposed men ("passive smokers"; average 72.8 hours exposure per week) were compared with 56 self-reported low ETS-exposed men (average 5.1 hours per week).[17] SCEs in cultured lymphocytes were examined; the authors reported that "[n]o difference was observed between low exposed non-smokers and the passive smokers." They concluded:

> Our results are in accordance with previous smaller studies in less homogeneous populations of non-smokers. These studies also failed to demonstrate even a tendency for an association between passive smoking and SCE levels. . . .

Five male smokers, five male nonsmokers, and five male nonexposed nonsmokers were compared in Holz and colleagues' 1993 paper.[1] The endpoint examined was DNA single-strand breaks (SSBs), "considered to be an important parameter of genotoxic stress," in lymphocytes. The authors write:

> All probands revealed measurable and varying SSB levels. Since DNA is an unstable molecule and estimated endogenous damage exceeds 100000 affected base pairs per cell per day, we assume that SSB base levels reflect unrepaired lesions. Active smoking caused an increase in SSBs in peripheral blood lymphocytes. This effect could not be found in passive smokers . . .

ETS exposure in this study consisted of five smokers each smoking 24 cigarettes in eight hours in an exposure chamber. This study provides no support for claimed genotoxic effects of ETS, even at a high exposure level.

Conclusion

This review of data from studies in which genotoxicity was assessed in persons actually exposed to ETS thus provides little, if any, support for the contention that ETS is genotoxic at levels encountered in workplaces and other indoor environments.

REFERENCES

1. Holz, O., Meissner, R., Einhaus, M., Koops, F., Warncke, K., Scherer, G., Adlkofer, F., Baumgartner, E., and Rudiger, H.W., "Detection of DNA Single-Strand Breaks in Lymphocytes of Smokers," International Archives of Occupational and Environmental Health 65: 83-88, 1993.

2. Ames, B.N., "Mutagenesis and Carcinogenesis: Endogenous and Exogenous Factors," Environmental and Molecular Mutagenesis 14 (Suppl. 16): 66-77, 1989.

3. Ames, B.N., and Gold, L.S., "Pesticides, Risk, and Applesauce," Science 244: 755-757, 1989.

4. Bombick, D.W., Ayres, P.H., Nelson, P.R., Coggins, C.R.E., France, D., Fulp, C., Lee., C., and Doolittle, D.J., "Assessment of the Biological Activity of Mainstream or Environmental Tobacco Smoke Using a Cellular Smoke Exposure Technique," Abstracts of the Twenty-Second Annual Scientific Meeting of the Environmental Mutagen Society, Kissimmee, Florida, April 6-11, 1991.

5. Sonnenfeld, G., and Wilson, D.M., "The Effect of Smoke Age and Dilution on the Cytotoxicity of Sidestream (Passive) Smoke," Toxicology Letters 35: 89-94, 1987.

6. Scherer, G., Westphal, K., Biber, A., Hoepfner, I., and Adlkofer, F., "Urinary Mutagenicity After Controlled Exposure to Environmental Tobacco Smoke," Toxicology Letters 35: 135-140, 1987.

7. Scherer, G., Westphal, K., Sorsa, M., and Adlkofer, F., "Quantitative and Qualitative Differences in Tobacco Smoke Uptake Between Active and Passive Smoking." In: Indoor and Ambient Air Quality. R. Perry and P.W. Kirk (eds.). London, Selper Ltd., 189-194, 1988.

8. Scherer, G., Westphal, K., Adlkofer, F., and Sorsa, M., "Biomonitoring of Exposure to Potentially Genotoxic Substances from Environmental Tobacco Smoke," Environment International 15: 49-56, 1989.

9. Scherer, G., Westphal, K., and Adlkofer, F., "Urinary Mutagenicity, Hydroxyphenanthrene, and Thioether Excretion After Exposure to Environmental Tobacco Smoke." In: Indoor Air Quality. H. Kasuga (ed.). Berlin, Heidelberg, Springer-Verlag, 138-146, 1990.

10. Scherer, G., Conze, C., Tricker, A.R., and Adlkofer, F., "Uptake of Tobacco Smoke Constituents on Exposure to Environmental Tobacco Smoke (ETS)," Clinical Investigator 70: 352-367, 1992.

11. Bos, R.P., Theuws, J.L.G., and Henderson, P.T., "Excretion of Mutagens in Human Urine After Passive Smoking," Cancer Letters 19: 85-90, 1983.

12. Mohtashamipur, E., Muller, G., Norpoth, K., Endrikat, M., and Stucker, W., "Urinary Excretion of Mutagens in Passive Smokers," Toxicology Letters 35: 141-146, 1987.

13. Bartsch, H., Caporaso, N., Coda, M., Kadlubar, F., Malaveille, C., Skipper, P., Talaska, G., Tannenbaum, S.R., and Vineis, P., "Carcinogen Hemoglobin Adducts, Urinary Mutagenicity, and Metabolic Phenotype in Active and Passive Cigarette Smokers," Journal of the National Cancer Institute 82: 1826-1831, 1990.

14. Collman, G.W., Lundgren, K., Shore, D., Thompson, C.L., and Lucier, G.W., "Effects of alpha-Naphthoflavone on Levels of Sister Chromatid Exchanges in Lymphocytes from Active and Passive Cigarette Smokers: Dose-Response Relationships," Cancer Research 46: 6452-6455, 1986.

15. Husgafvel-Pursiainen, K., "Sister-Chromatid Exchange and Cell Proliferation in Cultured Lymphocytes of Passively and Actively Smoking Restaurant Personnel," Mutation Research 190: 211-215, 1987.

16. Holz, O., Krause, T., Scherer, G., Schmidt-Preuss, U., and Rudiger, H.W., "^{32}P-Postlabelling Analysis of DNA Adducts in Monocytes of Smokers and Passive Smokers," International Archives of Occupational and Environmental Health 62: 299-303, 1990.

17. Gorgels, W.J.M.J., van Poppel, G., Jarvis, M.J., Stenhuis, W., and Kok, F.J., "Passive Smoking and Sister-Chromatid Exchanges in Lymphocytes," Mutation Research 279: 233-238, 1992.

SECTION XI

THE PROPOSED RULE ON ETS:

FEASIBILITY AND ALTERNATIVES

OSHA'S PROPOSED RULE ON ETS: FEASIBILITY AND ALTERNATIVES

OSHA HAS NOT DEMONSTRATED ANY CONTRIBUTION OF ETS TO CONSTITUENT LEVELS ABOVE BACKGROUND LEVELS IN THE WORKPLACE; OSHA HAS NOT MADE A DETERMINATION OF "SIGNIFICANT RISK" FOR AMBIENT ETS EXPOSURE LEVELS; OSHA FAILS TO PROVE THAT ETS CANNOT BE ADDRESSED THROUGH PROPER VENTILATION; OSHA FAILS TO EXPLORE ALTERNATIVES TO SMOKING BANS

OSHA states that the "primary objective of the tobacco smoke provision is to eliminate the nonsmoker's exposure to ETS. Under the Proposed Rule, firms will have the option of either banning smoking of tobacco products or permitting smoking only in designated areas." (59 FR 16016) The designated smoking area must be completely enclosed with a separate exhaust directly to the outside. In addition, the area must be negatively pressurized to prohibit exposure of any ETS constituent outside the designated area. (59 FR 16029) The Proposed Rule on ETS, according to OSHA, reduces "significant risk of material health impairment to the extent feasible." (59 FR 16013)

The Proposed Rule, however, does not explain why the complete elimination of ETS is required or how the studies that OSHA selected for its analysis of significant risk warrant the complete elimination of ETS. According to a recent court opinion (AFL-CIO v. OSHA, 965 F.2d 962, *975), OSHA's determination that a

2051817946

new standard is "reasonably necessary or appropriate" and that it "most adequately assures . . . that no employee will suffer material impairment of health or functional capacity, . . . necessarily requires some assessment of the level at which significant risk of harm is eliminated or substantially reduced." OSHA does not provide an assessment of the level of "significant risk of harm." That is because its analysis of significant risk for ETS is based upon two epidemiologic studies that are inconsistent with the body of evidence on health effects from workplace exposures to ETS, and from which estimated risks are generated without reference to actual ambient measures of ETS exposures.

OSHA provides no reason for setting its ETS standard at "zero" exposure. The zero level of exposure does not follow from OSHA's analysis of significant risk. It does not follow from OSHA's contention that its ventilation-based IAQ standard will not be effective in the removal and dilution of ETS constituents because "air exchange rates in non-industrial workplaces are not designed to control the risks of ETS exposure" (59 FR 15991), or because "the carcinogenicity of ETS discounts the use of general ventilation as an engineering control for this contaminant." (59 FR 15992) As discussed in Section IV of this submission, ordinary air in public places and workplaces contains many of the same substances imputed to ETS, in the complete absence of smoking.

Such substances, some of which are designated as "carcinogens" by OSHA, are produced by many common sources, and complete removal of ETS from the ambient air will not eliminate exposures to them. Moreover, some individual constituents found in both indoor air and ETS are already regulated by OSHA. OSHA establishes permissible exposure levels to such airborne substances presumably at levels protective of employee health and at which no significant risk of material impairment exists. The levels of exposure are typically far greater than any actually generated by ETS. OSHA's own Proposed Rule on IAQ does not call for the complete elimination of such substances, some of which are "carcinogens," but for the reduction of exposures to an "acceptable" level by ventilation.

OSHA thus has not demonstrated the extent to which ETS contributes to constituent levels above background levels in the workplace, it has not made a determination of "significant risk" for ambient ETS exposure levels, and it fails to prove that ETS cannot be addressed through proper ventilation, as spelled out in its own IAQ standard.

While the complete elimination of ETS is achievable through a smoking ban,* the complete elimination of the same

---

*. OSHA's rulemaking on ETS is directed at stopping the smoker rather than "protecting" the nonsmoker. A smoking ban with the express purpose of altering smoking behavior may be seen

(continued...)

constituents from other sources is not possible. If a comprehensive indoor air quality standard were truly the goal of the Proposed Rule on IAQ and ETS, then OSHA would have explored alternatives that are functionally equivalent in effectiveness to smoking bans in the reduction of exposures to ETS -- alternatives that would not seek to alter the behavior of the smoking workforce. A discussion of alternatives to smoking bans and/or separately ventilated smoking rooms follows below.

Current design and operation criteria for ventilation provide for the effective dilution and removal of ETS constituents from workplaces in which smoking is unrestricted; a ventilation Standard, ASHRAE 62-1989, has been incorporated into the major building codes in the U.S.; since 1990, the Standard has provided design and operations criteria for ventilation systems in new, remodeled and renovated buildings; the Standard has been incorporated by reference into OSHA's Proposed Rule on IAQ

The Proposed Rule states, without justification, that ventilation cannot be used to address the "risks" purportedly associated with exposures to ETS. The Proposed Rule specifically cites the "failure" of ASHRAE Standard 62-1989 in the "elimination" of "risks" from ETS exposures. (59 FR 15992) While ASHRAE Standard 62-1989 does not explicitly address purported "risks" from indoor air exposures, it establishes ventilation rates for various

---

*.(...continued)
as an attempt at "social engineering" and clearly is not, and ought not to be, within OSHA's regulatory framework.

locations in order to "control carbon dioxide and other contaminants with an adequate margin of safety, and to account for variations among people, varied activity levels, and a moderate amount of smoking" [emphasis added].[1] The Chairman of the ASHRAE Standards Project Committee for Standard 62-1989, Mr. John Janssen, writes:

> Laboratory research at Yale University and at the Technical University of Denmark has shown that 15 cfm of outdoor air will dilute occupant odors to a level acceptable to at least 80 percent of the people entering an occupied space from outdoors. Research on tobacco smoke odor at Yale's John B. Pierce Laboratory has also shown that with today's reduced smoking rate 15 cfm of outdoor air will dilute environmental tobacco smoke to a level acceptable to 70 percent of the people entering an occupied space where about 27 percent of the occupants smoke at a rate of 1.25 cigarettes per hour. This equates to one pack per 16 hour day. Other calculations on the tobacco smoke perception of nonsmoking occupants in a room for 15 minutes with smokers show that nonsmoking occupants will register 90 percent acceptance under the assumed conditions. Thus, Standard 62-1989 appears to be able to control tobacco smoke odor under minimum smoking conditions.[2]

The minimum ventilation rate recommended by ASHRAE Standard 62-1989, 15 cfm outside air/person, is the level at which odors, metabolic byproducts, and ETS are effectively diluted and removed.[3-5] (Ex. 3-440) The effectiveness of the minimum ventilation rate for the dilution of ETS has been evaluated by scientists.[6-8] In 1990, Pedelty and Holcomb demonstrated that air

quality in areas where smoking is permitted ad lib does not differ significantly from the quality of air in nonsmoking areas, where both areas are supplied with the minimum outdoor air ventilation rates recommended in ASHRAE 62-1989.[6]

In their review of ETS-related air quality monitoring in different workplaces under various smoking conditions, researchers from TDSA Ltd. conclude "in office areas in which (a) smoking is allowed and (b) outside air ventilation rates meet or exceed the ASHRAE Ventilation Standard, nicotine concentrations have typically been less than 5 ug/m^3 and respirable suspended particles have ranged between 20 ug/m^3 and 60 ug/m^3.[7] (Ex. 3-1073)

In their submission to the OSHA RFI Docket, scientists summarized the results of their paper on the measurement of ETS in 585 offices. (Ex. 3-1053) The authors write:

> Computer analysis shows that when "blind-folded" for presence or absence of smokers, in most cases realistic smoking levels do not significantly influence the aspects of air quality that were measured, and spillover from smoking areas into nonsmoking areas appears to be minimal. This work further reinforces the position the American Society of Heating, Refrigerating and Air-Conditioning Engineers has taken on ETS in office buildings in ASHRAE Standard 62-1989, that acceptable air quality can be maintained in properly ventilated offices with a moderate amount of smoking even without smoker segregation.

Professor Alan Hedge offers the following observation on the basis of his extensive experience in monitoring ETS constituents during investigations of sick-building syndrome: "Our data show that modern ventilation systems are capable of diluting the small pollutant loads from smoking at the levels which we observe, without necessarily exposing nonsmokers to significantly elevated levels of indoor air pollutants." (Ex. 3-955)

Company scientists from R.J. Reynolds (RJR) reported on a recently completed study of four office buildings.[8] (Ex. 3-1087) Two of the buildings investigated had a policy of unrestricted smoking and, in two other buildings, smoking was restricted to separately-exhausted lounges. Regardless of smoking policy, RJR reports that ventilation and indoor air quality indicators were "well within applicable standards." The authors write:

> In summary this study demonstrates conclusively (a) that with a HVAC system that is adequately designed, operated in accordance with current ASHRAE standards and properly maintained, all indicators for ETS are at extremely low, de minimis levels, even in the presence of substantial smoking activity, and (b) that such smoking activity has a negligible effect on contaminant levels in buildings where smoking is unrestricted.

They conclude:

> RJR believes, based on its own detailed research and the consistent results of other workplace assessments, that a properly designed and maintained HVAC system that is

operated in accordance with the ventilation rate procedures of the ASHRAE Standard 62-1989, will be effective in assuring that exposures to ETS will be de minimis.

Thus, in buildings meeting the ventilation rates specified in ASHRAE 62-1989, return air from areas in which smoking is permitted will be diluted by outside air, and the mixture of return and outside air will be filtered prior to returning to the supply system. The dilution factor accounts for the low levels of ETS constituents measured in nonsmoking areas, as documented in the above studies.

Simple physical separation of smokers and nonsmokers has been effective in the reduction of nonsmoker exposure to ETS; simple spatial separation of smokers and nonsmokers, even under conditions of recirculated ventilation, effectively minimizes ETS exposure for nonsmokers; data do not support a significant reduction in ETS exposures beyond adequate ventilation and/or simple separation of smokers and nonsmokers

Scientific studies indicate that the simple physical separation of smokers and nonsmokers, even under a shared ventilation system with recirculated air, can effectively minimize nonsmoker exposures to ETS.[7,9-20] Other studies indicate that smoking bans and/or separately ventilated smoking areas do not significantly reduce ETS exposures beyond reductions achieved through simple separation and/or adequate ventilation.[8,9,12-14,17]

A 1990 study by Vaughn and Hammond, cited in the Proposed Rule (59 FR 15991), examined the impact of smoking policies on ETS constituent levels in a high-rise building.[9] The authors reported an 80 percent reduction in exposure to ETS constituents in nonsmoking workareas after designation of a smoking area on a floor that utilized a common (recirculating) air-handling system. Exposure levels to nicotine in nonsmoking workareas prior to the designation of the smoking area were 2.0 ug/m^3; after institution of the smoking policy, nicotine levels dropped to 0.1-0.3 ug/m^3. A complete smoking ban on another floor in the building produced a 95+ percent reduction in ETS constituents, only a 15 percent exposure reduction beyond simple separation of smokers and nonsmokers.[9]

Another study of a smoking-restricted office building reported that ambient nicotine in nonsmoking areas was virtually undetectable.[10] Smoking was restricted to designated areas with local air filtration systems. The authors concluded that spatial restrictions are "effective in minimizing the impact of environmental tobacco smoke on indoor air quality."[10]

In a similar study, Canadian researchers compared measured ETS constituents in offices where smoking was regulated and unregulated.[7,11] They reported no significant differences in average ETS constituent levels between nonsmoking offices that

received recirculated air from designated smoking areas, and nonsmoking offices that did not receive recirculated air. Nicotine concentrations reported for nonsmoking areas were only marginally above limits of detection and quantitation; there were no measurable differences in particles or carbon monoxide levels in nonsmoking areas that did or did not receive recirculated air from smoking areas. The researchers concluded:

> The results indicate that the provision of a designated, but not separately ventilated, smoking area can effectively eliminate or drastically reduce most components of environmental tobacco smoke for nonsmoking offices.[11]

In 1991, Hedge, et al. examined the effects of smoking policies on indoor air quality in 18 private-sector buildings.[12] The study covered over 3,000 workers. They concluded:

> Comparison of all open-office sites between policies showed no significant differences in levels of carbon monoxide, carbon dioxide, formaldehyde or respirable particulates. . . . Smoking policy had a relatively small effect on IAQ for the pollutants measured. For most of these pollutants, there were no significant differences in concentrations among offices in SP (smoking-prohibited) buildings, nonsmoking office areas in RF (smoking restricted to rooms with local filtration), RSV (smoking restricted to rooms with separate ventilation) and RMP (smoking restricted to rooms with no location air treatment) buildings, and office areas in RWS (smoking restricted to open-plan cubicle workstations and enclosed office) buildings. There was a significant effect of smoking policy on UVPM and formaldehyde in these office areas . . . however, all

concentrations of UVPM and formaldehyde were low.

A 1993 follow-up study by the same authors compared ETS constituent levels in 27 office buildings under five different kinds of smoking policies.[13] The smoking policies ranged from unrestricted smoking to the complete prohibition of smoking. The authors report that nicotine and tobacco-specific particles (UVPM-RSP) were measurable in offices that permitted smoking, but exposures to other airborne substances were similar across all buildings, regardless of smoking policy. The authors estimated that a typical nonsmoker would be exposed to the nicotine equivalent of approximately three cigarettes per year in open-plan offices with smoking restricted to enclosed parameter offices. Simple separation of smokers and nonsmokers under a common ventilation system was estimated to result in nicotine exposure levels of no more than five cigarette equivalents per year.

A 1993 Canadian study compared exposures to ETS constituents in three buildings before and after smoking bans.[14] The authors reported a significant reduction in average levels of volatile organic compounds in the buildings after the smoking ban, a result they could not explain and one that is inconsistent with other studies that demonstrate no significant contributions from ETS to indoor levels of total volatile organic compounds.[15,16] The smoking ban, however, had no significant effect on overall

exposures to carbon monoxide or particles, or on cotinine levels in body fluids of nonsmokers.

Similarly, Proctor (1987) monitored ETS constituents before and after a smoking ban on public transportation in the United Kingdom.[17] While nicotine concentrations decreased from 7 ug/m^3 to 3 ug/m^3 in nonsmoking compartments after the ban, particulate and carbon monoxide levels remained unchanged. This suggests that ETS contributions to levels of particulate and CO in public transportation are not significant.

In another study by Proctor and co-workers (1989), the researchers measured nicotine, RSP, carbon monoxide, carbon dioxide, and volatile organic compounds in the air of smokers' and nonsmokers' offices.[15] The data indicate little nonsmoker exposure to various ETS constituents through simple separation. The average UVPM-RSP level in nonsmokers' offices was 8.8 ug/m^3; the median nicotine value was less than 1 ug/m^3. Carbon monoxide and carbon dioxide levels did not differ appreciably between smokers' and nonsmokers' offices. Overall, levels of volatile organic compounds did not differ significantly between smokers' and nonsmokers' offices.

Bayer and Black (1987) reached a similar conclusion in their investigation of volatile organic compound levels in smokers

and nonsmokers' offices.[16] They noted that although differences in nicotine concentrations were measurable for offices of smokers compared with nonsmokers, no significant differences in volatile organic compounds were discerned in smokers' and nonsmokers' offices. The researchers observed that "it was not possible" to correlate volatile organic compounds with ETS or to attribute the source of various volatile organics to ETS.

A 1989 study performed for the U.S. Department of Transportation on ETS constituent levels aboard commercial aircraft indicates the overall effectiveness of simple separation of smokers and nonsmokers in the minimization of ETS exposures.[18] This study is cited in the Proposed Rule. (59 FR 15991) The researchers reported an average level of 0.11 ug/m^3 nicotine in nonsmoking sections for their sample of 61 domestic commercial flights. The average level was over 100 times lower than that measured in smoking sections; it is equivalent to 1/8000 the nicotine delivery of a single cigarette.

In the largest study of its kind, researchers reported measurements of ETS constituents in 585 offices, many of which were conventional office settings with simple separation of smokers and nonsmokers under common air-handling systems and recirculated air.[19] The researchers concluded that: "[I]n most cases realistic smoking levels do not significantly influence the aspects of air quality

that were measured, and spill-over from smoking areas to nonsmoking areas appears to be minimal." "Spill-over" of tobacco smoke constituents was reported in only four percent of the nonsmoking areas.

Lambert, et al. (1993) examined differences in nicotine and RSP levels in the nonsmoking and smoking sections of restaurants.[20] Simple separation of smokers and nonsmokers in restaurants resulted in substantial reductions of exposure to RSP and nicotine for nonsmokers. Nicotine concentrations averaged 65 percent lower in nonsmoking sections than in smoking sections; RSP concentrations were 40 percent lower. The average concentration of nicotine in smoking areas was 3.2 ug/m^3 compared with 1.0 ug/m^3 in nonsmoking areas. The difference between average RSP levels in smoking and nonsmoking sections was 26 ug/m^3, a level consistent with those reported for the contribution of ETS in homes and in offices with smokers. (See Table V, Section IV)

The studies reviewed above contain data regarding the low levels of ETS constituents in nonsmoking areas under conditions of simple separation of smokers and nonsmokers with recirculation of ventilation air. Data reported in those studies indicate that ETS constituent levels in nonsmoking areas in buildings where smoking is permitted are often only slightly above limits of detection and quantitation, and often statistically indistinguishable from

"background" levels of such constituents found in buildings in which smoking is altogether prohibited. The data support the contention that simple physical separation of smokers and nonsmokers effectively reduces and minimizes ETS exposure in nonsmoking areas, even under conditions of recirculated ventilation.

There are substantial data, submitted to the OSHA RFI docket on IAQ and reviewed in this section, that indicate that typical workplace exposures to ETS constituents are low and reducible to de minimis levels through the simple physical separation of smokers and nonsmokers in conjunction with the current ventilation rates adopted in OSHA's Proposed Rule on IAQ. **The Proposed Rule provides no discussion or scientific data that would support a finding that ETS is related to any material health impairment at exposure levels encountered through simple separation and/or adequate ventilation.**

Negative air pressure zones; if physical grouping of smokers and nonsmokers in discrete areas is desired by employers, prevailing air circulation currents and routes of supply and exhaust air should be considered; if possible, smoking areas should be placed near existing exhausts so that air movement will be directed from nonsmoking to smoking areas, thereby minimizing possible migration of tobacco smoke from smoking areas

According to Hayward, et al. (1993), the effectiveness of a designated smoking area for controlling exposure to ETS in nonsmoking areas is determined by two basic factors.[21] The first requires the successful dilution and removal of ETS constituents within the smoking area. The ventilation rate, either through outside air or transfer air (the air from other zones within the building), is the most critical determinant for the dilution and removal of ETS constituents. Outside air should be supplied at rates designated by ASHRAE Standard 62-1989.

A second factor related to the movement of ETS constituents from smoking areas to nonsmoking areas depends upon airflows within the structure.[21] Airflow is affected by the existence of physical barriers such as walls or partitions, as well as by air pressure relationships within the building. For a designated smoking area without partitions, the simple location of smokers near existing exhausts and the designation of nonsmoking areas near supply air diffusers will prevent movement of ETS constituents into nonsmoking areas. Airflow will be directed from

the nonsmoking or supply air areas into smoking areas, thereby preventing the air from the smoking area from re-entering that of the nonsmoking area. This technique, known as "air pressure zoning," has been described in a recent publication for design engineers:

> Air generally flows from areas of higher air pressure to areas of lower air pressure, from positive pressure in the direction of negative pressure. Using this simple concept, areas set aside for nonsmokers can be maintained at a slight relative positive pressure, while areas set aside for smokers can be maintained at a slight negative relative air pressure. This will produce a slight airflow from the nonsmoking area into the smoking area, keeping the air from the smoking area from mixing with that of the nonsmoking area. Through thoughtful planning and carefully supervised and tested balancing of the HVAC system, the preferences of both smokers and nonsmokers can be accommodated without any additional cost to building operations.[22]

Air pressure zoning involves the use of existing ventilation systems, e.g., supply and exhausts within a building, and will not influence capital costs or operating efficiencies for a building.

An alternative to the separate smoking area required in the Proposed Rule (i.e., an enclosed space with dedicated exhaust under negative pressure) utilizes the theory of negative pressure and the use of transfer air, which is air drawn directly from other parts of an occupied space. The use of transfer air in a practical

smoking lounge design is permitted under ASHRAE Standard 62-1989. The lounge would be ventilated in a way similar to the way restrooms are ventilated and exhausted. Restrooms in public buildings are equipped with exhaust ventilation for the removal of odors, etc. The restroom often draws its supply air from adjacent areas such as corridors that are close to the restroom. The rooms adjacent to the restroom are not fitted with comparable exhaust capabilities. The exhaust air from the restroom creates a negative pressure relative to its adjoining areas. Air is thus "transferred" from adjacent areas of positive pressure into the restrooms and, if the exhaust is working properly, the result is the creation of a negative pressure zone.

The 1993 publication by Hayward, et al. examined the effect of negative pressurization on movement of ETS constituents in three separate buildings.[21] In one of the buildings, the use of a small exhaust fan in the smoking area dramatically reduced migration of ETS constituents into nonsmoking areas. Exposures to nicotine and RSP in nonsmoking areas were reduced below the limit of quantitation. In a second building, the effects of negative pressurization were less dramatic because the ventilation was very effective from the outset in removing and diluting ETS constituents. A third building was not negatively pressurized in smoking areas and nonsmoker exposure to ETS constituents was greater than in the other two buildings.

In 1993, Light and Gay measured nicotine levels in two office buildings with a variety of areas designated for smoking.[23] Nicotine was below the level of detection (less than 0.7 ug/m^3) in most of the sites measured. The authors concluded: "Within the sensitivity of the tests and observations performed, exposure was not documented from the recirculation of air even though many smoking areas were not exhausted to the outside. This suggests that there was little, if any, hazard under the conditions evaluated in areas potentially receiving recirculated ETS but not immediately adjacent to smoking." They reported that "positive pressurization of smoking rooms" led to "intermittent nonsmoker exposure in immediately adjoining areas." If the smoking areas were negatively pressurized, no detectable exposure to ETS constituents occurred adjacent to smoking areas.

Feasibility of OSHA's Proposed Rule on ETS

According to OSHA, "the primary objective of the tobacco smoke provision [of the Proposed Rule] is to eliminate the nonsmoker's exposure to ETS." (59 FR 16016) OSHA further states: "Under the Proposed Rule, firms will have the option of either banning smoking of tobacco products or permitting smoking only in designated areas." (59 FR. 16016) The Proposed Rule requires that designated smoking areas be enclosed, exhausted directly to the outside and maintained under negative pressure. (59 FR 16032)

Under OSHA's Proposed Rule for ETS, constituents imputed to ETS will be "eliminated" from the workplace, while exposures to the same constituents from other sources will be minimized to presumably acceptable levels by the ventilation-based Proposed Rule for IAQ. The foregoing analysis clearly demonstrates that: (1) ETS constituent levels in typical workplaces are low and nonsmoker exposure to ETS constituents is minimal; (2) simple physical separation of smokers and nonsmokers in the workplace provides for significant reductions of already minimal exposures to ETS constituents; (3) adequate ventilation effectively dilutes and removes ETS constituent levels to the extent that levels will often fall below levels of detection or quantitation and will not differ significantly from background levels of constituents generated by other sources; and (4) the negative pressurization of smoking areas will prevent "migration" or "spillover" of ETS constituents into nonsmoking areas.

OSHA's proposal to completely eliminate ETS constituents is a regulation that seeks to modify already insignificant levels of ETS exposure. The alternatives described above were not considered by OSHA, yet they are equivalent in effectiveness to OSHA's proposed requirement of a separately enclosed, separately exhausted, and negatively pressurized smoking room. **The Proposed Rule will produce only trivial and insignificant reductions in**

exposures to ETS constituents over the alternative provisions specified above.

OSHA's Proposed Rule on ETS ostensibly provides a choice regarding smoking for the nation's employers: either ban smoking or construct special smoking rooms. However, the choice is not real. The Proposed Rule constitutes a de facto ban on smoking because OSHA trivializes and minimizes the economic and technological feasibility of providing separate rooms for smoking employees. The "option" of providing a separately ventilated smoking room lies with employers (not building owners), even when the employer leases space for his or her business. OSHA clearly recognizes this impossible scenario in its Proposed Rule, e.g., "since changes in building ventilation systems will be made by landlords, employers may have to negotiate agreements to ensure that they can meet the OSHA Standard. On the requirement for ETS, landlords in turn are likely to pressure employers to ban smoking; thereby forestalling any need for construction of designated smoking rooms." (59 FR 16013)

OSHA declares that problems concerning the technological feasibility of the Proposed Rule "are not evident." (59 FR 16013, 16023) However, the isolation of smokers in a separate room as required by OSHA's Proposed Rule places additional demands on an existing ventilation system. The Proposed Rule's requirement of a

separate exhaust leading directly to the outdoors is not feasible in many buildings.[22] Few existing buildings, particularly high-rise buildings, are amenable to providing exhaust directly to the outdoors from any given location within the building. The option is not technologically feasible in these instances, and the employer, under OSHA's Proposed Rule, would have no choice but to completely ban smoking. The Proposed Rule concedes: "OSHA recognizes that not all establishments will make available designated smoking areas as there may be physical constraints on the option of providing separate ventilation. Such constraints are imposed by the building's design, the building's mechanical ventilation system's capabilities, by cost involved in providing adequate ventilation, by the occupant use of the building." (59 FR 16016) The U.S. EPA recently conceded that "the structural features of many existing buildings make it infeasible or cost prohibitive to construct a smoking lounge" similar to that envisioned by OSHA.[24] The EPA report suggests that smoking lounges would be feasible in only 10 to 20% of the existing buildings in the U.S. Tenants in 80 percent or more of existing buildings would be forced to ban smoking altogether under OSHA's Proposed Rule.

OSHA has placed the burden of its Proposed Rule on ETS upon the nation's employers by presenting them with a "choice" over which they are not empowered (i.e., to ban smoking or restrict it to a specially ventilated room). For the employer who is not also

a building owner, there is no real choice in the matter. For either the tenant or owner of a multi-story building, there may be no "choice" in the matter due to feasibility restrictions. For the small business owner who must lease additional space for a smoking lounge, there may be no "choice" in the matter. For other businesses, retrofit requirements for the construction of an enclosed, separately exhausted and negatively pressurized smoking room may be cost-prohibitive, and the "choice" in the matter disappears. The Proposed Rule does not address these issues.

The minimum outdoor air ventilation rates required in OSHA's Proposed Rule on IAQ are based on versions of ASHRAE Standard 62 (for ventilation); the current ventilation standard, ASHRAE 62-1989, provides minimum outdoor air ventilation rates for ETS; the precursor standard, 62-1973, specified recommended ventilation rates comparable to ASHRAE 62-1989 and served as the ventilation basis for building codes since 1973; OSHA's ventilation recommendation for IAQ will therefore adequately address ETS; OSHA's separation of ETS from general IAQ has no basis

OSHA's Proposed Rule for IAQ (to the exclusion of ETS) states that "employers [must] maintain and operate the HVAC system to provide at least the minimum outdoor air ventilation rate, based on actual occupancy, required by the applicable building code, mechanical code, or ventilation code in effect at the time the facility was constructed, renovated, or remodeled, whichever was most recent." (59 FR 16026-27) In the foregoing analysis, it was

demonstrated that, contrary to OSHA's contention regarding the inapplicability of ventilation to ETS, ASHRAE Standard 62-1989 for Ventilation was specifically designed for, and has been proven successful in, the dilution and removal of ETS constituents. The Standard currently provides ventilation design/operation criteria for building codes in the U.S.

A precursor standard to ASHRAE 62-1989, ASHRAE Standard 62-1973, recommended a ventilation rate of 15 cfm outside air/person, comparable to the minimum recommended outdoor air rate specified in ASHRAE 62-1989. Standard 62-1973 was approved by the American National Standards Institute (ANSI) and incorporated into building codes. It was in effect through the 1970s. ASHRAE updated Standard 62-1973 in 1981 and provided for two ventilation rates based on smoking and nonsmoking (Standard 62-1981). That Standard did not receive approval from ANSI and was not incorporated into the major building codes in the U.S. ASHRAE 62-1973 thus remained in effect throughout the 1980s as the design and operational criteria document for ventilation in building codes. Many HVAC systems designed and installed over the past 20 years have complied with the specifications in ASHRAE 62-1973 and ASHRAE 62-1989.

The ASHRAE Standards (62-1973 and 62-1989) designate the required outdoor air portion of total supply air, where total

supply air equals outside air and return air. The minimum outdoor rates specified in the Standards actually constitute only a fraction of the supply air needed to provide proper heating and cooling; outside air supply requirements constitute a small percentage of the air needed for total supply air. If outside air specifications differ from Standard to Standard, overall HVAC capacities, as determined by minimum design criteria for supply air, would satisfy the demand for greater outdoor air flow rates. Indeed, recent research on 160 office buildings by Sundell, et al. indicated that buildings of different age categories did not differ operationally with regard to outdoor air flow rates.[25] The assumption that an increase in outside air ventilation rates would require the redesign and retrofit of an existing ventilation system is not supported.

Differences in minimum outside air requirements between the two versions of the ASHRAE Standard are not likely to be significant and may, in fact, be identical for certain indoor areas. **Thus, compliance with the Proposed Rule's requirements for minimum outdoor air ventilation rates for IAQ, insofar as they are based on the 1973 and 1989 versions of the ASHRAE Ventilation Standard, would be sufficient to satisfy the ventilation requirements for the effective dilution and removal of ETS constituents under ad lib smoking situations.** There are no feasibility constraints based on compliance with current building

code specifications for ventilation, as specified in OSHA's general ventilation recommendation for IAQ.

As OSHA's Proposed Rule on ETS tacitly admits, an enclosed, separately exhausted and negatively pressurized smoking room is neither technologically nor economically feasible as a real option available to all of the nation's employers. (59 FR 16016, 16013) As demonstrated above, negative pressurization is feasible using existing exhaust and supply locations in a building. A requirement for a separate exhaust from a smoking area directly to the outside is burdensome, superfluous and dictated only by OSHA's "zero exposure" doctrine for ETS. Scientific studies and IAQ reports indicate that smoking and nonsmoking areas may share a common air handling system with recirculation of air such that constituents from ETS from smoking areas are not significantly redistributed to nonsmoking areas. This condition can be achieved if the building conforms to the specifications for ventilation rates and filtration recommended by the current ASHRAE Standard for ventilation -- indeed, by OSHA's own Proposed Rule on IAQ.

REFERENCES

1. ANSI/ASHRAE Standard 62-1989, "Ventilation for Acceptable Indoor Air Quality," American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc., ASHRAE, Atlanta, Georgia, 1990.

2. Janssen, J., Statement of ASHRAE, U.S. House Energy and Commerce Subcommittee on Health and the Environment, Washington, D.C., April 10, 1991.

3. Goodfellow, H., and Wyatt, J., "Critical Review of Ventilation Contaminants in Indoor Air." In: Indoor Air Quality and Ventilation. F. Lunau and G. Reynolds (eds.). London, Selper Ltd., 95-104, 1990.

4. Binnie, P., "The Role of Ventilation in Controlling the Quality of Indoor Air, Including ETS." In: Other People's Tobacco Smoke. A. Armitage (ed.). Beverly, East York, United Kingdom, Galen Press, 159-172, 1991.

5. Burroughs, H., "ASHRAE: Addressing the Indoor Air Quality Challenge With Energy Conscious Design." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 530-535, 1988.

6. Pedelty, J., and Holcomb, C., "A Computer Simulation of Indoor Air Quality Which Models Changes in Point Sources and Ventilation," Environmental Technology 11: 1053-1062, 1990.

7. Sterling, T., and Mueller, B., "Concentrations of Nicotine, RSP, CO and CO_2 in Non-smoking Areas of Offices Ventilated by Air Recirculated From Smoking Designated Areas," Am Ind Hyg Assoc J 49(9): 423-426, 1988.

8. Oldaker, G., et al., "Investigations of Ventilation, Smoking Activity and Indoor Air Quality at Four Large Office Buildings," IAQ '92: Environments for People, Atlanta, ASHRAE, 1992: 248-257.

9. Vaughan, W., and Hammond, K., "Impact of 'Designated Smoking Area' Policy on Nicotine Vapor and Particle Concentrations in a Modern Office Building," J. Air Waste Manage. Assoc. 40: 1012-1017, 1990.

10. Hedge, A., et al., "Building Ventilation and Smoking Policy Effects on Indoor Air Quality and Employee Comfort and

Health," Indoor Air '90, Toronto, Canada, July 29-August 3: 739-744, 1990.

11. Sterling, T., and Mueller, B., "ETS in Offices and When Smoking is Restricted to Designated but Not Separately Ventilated Areas." In: Indoor Air Quality. H. Kasuga (ed.). Springer-Verlag, Berlin, Heidelberg: 120-129, 1990.

12. Hedge, A., et al., "The Effects of Smoking Policy on Indoor Air Quality and Sick Building Syndrome in 18 Air-Conditioned Offices," IAQ 91: Healthy Buildings: 151-159, 1991.

13. Hedge, A., et al., "Effects of Restrictive Smoking Policies on Indoor Air Quality and Sick Building Syndrome: A Study of 27 Air-Conditioned Offices," Indoor Air '93 (1): 517-522, 1993.

14. Broder, I., et al., "Environment and Well-Being Before and Following A Smoking Ban in Office Buildings," Can J Pub Health, 84(4): 254-258, 1993.

15. Proctor, C., et al., "Measurements of Environmental Tobacco Smoke in an Air-Conditioned Office Building," Environ Technol Letters (10): 1003-1018, 1989.

16. Bayer, C., and Black, M., "Thermal Desorption/Gas Chromatographic/Mass Spectrometric Analysis of Volatile Organic Compounds in the Offices of Smokers and Nonsmokers," Biomed and Envir Mass Spect 14(8): 363-367, 1987.

17. Proctor, C., "A Study of the Atmosphere in London Underground Trains Before and After the Ban on Smoking," Toxicology Letters, Proceedings of the International Experimental Toxicology Symposium on Passive Smoking, ed., E. Mohtashamipur, Amsterdam, Elsevier, 131-134, October 23-25, 1986.

18. Nagda, N., et al., "Airliner Cabin Environment: Contaminant Measurements, Health Risks and Mitigation Options," Geomet Technologies Final Report, U.S. Department of Transportation, 1989.

19. Turner, S., et al., "The Measurement of Environmental Tobacco Smoke in 585 Office Environments," Environment International 18: 19-28, 1992.

20. Lambert, W., et al., "Environmental Tobacco Smoke Concentrations in No-Smoking and Smoking Sections of Restaurants," Am J Pub Health 83(9): 1339-1341, 1993.

21. Hayward, S., et al., "Effectiveness of Ventilation and Other Controls in Reducing Exposure to ETS in Office Buildings," Proceedings of Indoor Air '93, 5: 509-514, 1993.

22. Benda, G., "The New Horizon: Indoor Environmental Quality," Consulting-Specifying Engineer (Invironment) Supplement: June, 1993: 82-104.

23. Light, E. and Gay, R., "Preliminary Assessment of Designated Smoking Areas for Non-Smoker Exposure to Environmental Tobacco Smoke," Indoor Air Quality '93: Operating and Maintaining Buildings for Health, Comfort and Productivity, ASHRAE, Philadelphia, Pennsylvania, November 7-10, 1993. Presentation.

24. Mudarri, D., "The Costs and Benefits of Smoking Restrictions: An Assessment of the Smoke-Free Environment Act of 1993," U.S. EPA, Office of Radiation and Indoor Air, Indoor Air Division 6607J, April, 1994.

25. Sundell, J., et al., "Associations Between Type of Ventilation and Flow Rates in Office Buildings and the Risk of SBS-Symptoms Among Occupants," Environment International 20(2): 239-251, 1994.